cholinergic reflex arc. This hypothesis is especially attractive because it is known that inhaled sulfur dioxide activates tracheobronchial irritant receptors that in turn stimulate the afferent arc of the cholinergic reflex. All sulfites are converted to sulfur dioxide in solution. The mechanism of susceptibility to metabisulfites in these patients, however, remains obscure.

Metabisulfite sensitivity probably is not rare. A history of anaphylaxis occurring following ingestion of restaurant food and drink, particularly salads and avocado dip, suggests that metabisulfite sensitivity reactions have occurred. Anaphylaxis after ingestion of soft drinks with sulfur dioxide or wines; shrimp or other seafood, or restaurant potatoes or vegetable combinations sprayed with potassium metabisulfite solutions should alert a physician to the possibility of this syndrome.

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Urticarial Vasculitis

URTICARIA IS CHARACTERIZED clinically as pruritic, erythematous, cutaneous elevations arising from dilated blood vessels and areas of cutaneous edema. After intradermal injection, many mediators of inflammation are capable of producing lesions that resemble urticaria. These mediators include histamine, the anaphylatoxins of complement (C3a, C4a and C5a), certain leukotrienes, some prostaglandins and perhaps serotonin.

Inflammation within blood vessels is called vasculitis and may appear as the consequence of one or more mechanisms. As detected by routine histologic and immunofluorescence techniques, vasculitis is frequently found in acute urticaria precipitated by adverse drug reactions, hepatitis B infection or serum sickness due to heterologous protein administration. Vasculitis is less frequently observed in the specimens from skin biopsies of patients with chronic idiopathic urticaria. However, recent investigations clearly show characteristic histologic or plasma complement abnormalities, or both, in some patients with chronic idiopathic urticaria.

Although mild inflammatory changes of vasculitis are detected in the specimens from skin biopsies of many patients with chronic idiopathic urticaria, the patients with chronic urticaria and necrotizing venulitis and others with hypocomplementemic vasculitic urticarial syndrome constitute distinct subsets of urticarial patients with more prominent systemic features and more severe vasculitis. Necrotizing venulitis was observed in adults with recurring urticaria, episodic arthralgias or arthritis, abdominal pain or glomerulitis. The plasma complement analyses in some of these patients indicated activation of the classical pathway of complement as shown by decreased total hemolytic complement (CH₅₀), Clq, C4 and occasionally C3. In other patients the complement analyses were normal.

The other syndrome, called the hypocomplementemic vasculitic urticarial syndrome or systemic lupus erythematosus (SLE)-related syndrome, was observed in adult women who have persistent urticaria, leukocytoclastic angiitis, severe angioedema, arthralgias, arthritis, neurologic complications, sensitivity to potassium iodide and profound hypocomplementemia. The complement analyses of these patients' plasmas showed depressed CH₅₀, C4, C2 and C3 in association with deficiency of Clq. The deficient Clq apparently relates to the presence of an unusual immunoglobulin that precipitates Clq (7S Clq precipitin or low molecular weight Clq precipitin). Both the Clq deficiency and the 7S Clq precipitin are characteristic of this syndrome.

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Rhinitis

INFLAMMATION OF THE nasal mucosa is commonly termed rhinitis. There are a number of causes. A diagnostic classification can be based on more specialized nasal evaluations including nasal cytology, rhinomanometry, nasal challenge, and olfactory and ciliary function testing.

Nasal cytology is carried out by gently scraping the mucosal lining of the inferior turbinate with a calcium-alginate applicator and placing the specimen on a glass slide. The prepared slide is then metachromatically stained and the cell pattern examined microscopically. Nasal airway resistance